Chapter 33

Occupational Chest Diseases

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Permanent disability resulting from occupational factors always has been and still remains an important problem in industrial medicine. In the United States alone more than 4 million industrial workers are exposed to potentially hazardous substances. The current bibliography on the subject exceeds 8,000 references, and it is obviously possible to review here only those occupational chest ailments which are of greatest topical importance. At the same time, emphasis will be placed on basic principles and pragmatic issues.

I. SILICOSIS

Because of the predominance of silica in the earth's crust, it is natural that silicosis should constitute an important occupational chest disease. Of the more than 3,000 known minerals, more than 500 are compounds of silica. It is indeed fortunate that many of these naturally occurring varieties of silica, as well as the element silica, are biologically inert. It should, however, be cautioned that epidemiologic surveys and experimental inquiry have concerned themselves with fewer than 10 per cent of these substances. In recent years, numerous synthetic siliceous substances have been introduced.

Many theories of the biological action of SiO_2 have been suggested, *e.g.*, *(a)* the mechanical injury concept, which presumed that angular quartz particles can lacerate vital cell components; *(b)* the solubility theory, silicic acid being presumed to be the pathogen; *(c)* the polymerization theory, the slow formation of polysilicic acid being invoked to explain the retarded development of silicotic nodules; *(d)* the piezoelectric theory, based on the assumption that the well-known piezoelectric forces characteristic of quartz may cause local tissue injury; *(e)* the unsaturated valency theory, which postulates the existence of a layer of highly reactive SiO molecules at the fracture angles of quartz particles; *(f)* the colloidal silica theory, which is based on the concept that the crystalline quartz particle is covered with a film of toxic colloidal silica; *(g)* the protein denaturing theory, which postulates that the physical field at the surface of the quartz particle induces organic chemical

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problem. Some dusty trades are not, however, readily a engineering control. Mines are notorious in this respect a continue to contribute many new cases. Even in such in prevalence rates, speed of progression, incidence of compl life expectancy have been favorably modified by partial dust pre-employment and periodical medical examinations, isolat culous subjects and earlier exclusion of silicotic workers from areas.

Aluminum inhalation has been added as a valuable adj industries where dust suppression cannot be achieved. Use tically, submicron oxidized metallic aluminum inhaled a for fifteen minutes twice daily, before and after the work prevented or retarded silicosis in some 40,000 miners, fou and ceramic industry employees whose health would have serious jeopardy because of prevailing dust exposures. Alu ation has also proved valuable in mitigating, retarding or p progression of established silicosis. The validity of this met established by extensive animal experimentation, and the attendant on its proper use.

No other known medicinal aids, other than palliative Generally, it is better to keep the silicotic physically acti possible in order to retard fibrotic contraction of the lung. sema, tuberculosis, or cor pulmonale have supervened, s in physical exertion becomes necessary. Complete bed often hastens the patient's demise. The decision as to w follow is often very much a Hobson's choice.

II. SILICATOSES

While the foregoing discussion has been concerned p the disease produced by inhaled silicon dioxide, the silica extensive array of naturally occurring minerals widely use With the exception of the fibrous silicates, the majority o are relatively inert, exciting little more pulmonary reactio cytosis. This is the reason for stressing the importance combined silica. Some of the silicatoses can, however, l the most severe case of silicosis.

Asbestosis: This is by far the most important among Six varieties of asbestos fibers are commonly implicated, amosite, crocidolite, anthophyllite, aktinolith and tremoli than 85 per cent of the asbestos used in the U.S.A. is c occurring in this country are most probably chrysotile-der lite is more commonly mined and used in Europe and information leaves scope for precise identification of the has caused the asbestosis since the different types of asb be distinguished in biopsy or autopsy material.


A
B

FIG. 56

ASBESTOSIS

HISTOPATHOLOGY

A. DOMINANTLY SUBPLEURAL ASBESTOSIS. The main zone of collagen deposition is in the subpleural zone on either side of the lamina elastica. Fibrous trabeculae dip inward along venous channels. Dilated bronchi within zones of condensation. Dispersed fine focal perivascular fibrosis. *Miner exposed to chrysotile dust 23 years.* Initially symptomless. Died of cor pulmonale.

B. ADVANCED ASBESTOSIS. Dense linear perivenous fibrosis with minimal vesicular emphysema. *Textile worker exposed to amosite dust 11 years.* Progressive exertional dyspnea. No cough. Died of cor pulmonale.

C. TALCOSIS. Irregular interstitial fibrosis, mainly perivenous and peribronchial. Marked distension of lesser bronchi and bronchioles. *Mill worker exposed to tremolite talc dust 17 years.* Terminal progressive dyspnea and productive cough. Died of cor pulmonale.

Asbestosis is provoked wherever exposures to asbestos dust occurs. However, in textile industries there appears to be a greater prevalence of severe disease. In contrast with silicosis, the asbestotic reaction in the lung is excited not by minute particles but by relatively long fibers. Thus, if asbestos be pulverized to particles no longer than 3 microns, very little disease develops and the process advances much more slowly than when the aerosol contains a predominance of fibers of 10 to 50 microns. Again, if the fibers are introduced into the lung in a high caliber form (*e.g.* conjoined multiple fibrils), less disease results than when the individual fibers are split into their component fibrils of submicron caliber.

Asbestosis is essentially an interstitial pulmonary lesion in which all component tissues are involved with focal emphasis of the process. The lesions range in severity from mere alveolar mural cellular infiltration of a microscopic nature, not detectable by radiography or even on gross anatomical examination, to massive consolidation with associated vascular occlusion, bronchiectasis, and carcinomatosis.

The interstitial lesions result after fine short fibers have been ingested by phagocytes and transferred into the lymphatic channels of the alveolar septa. Here fibrocytes proliferate, new capillaries form and reticulin, collagen and new elastic fibers are laid down. This type of lesion cannot readily be distinguished from other interstitial pneumonitides unless the asbestos fibrils are detected by oil immersion or electron microscopy. Rarely, some of these fibrils will be rendered obvious by conversion into an asbestos body through proteinaceous encapsulation and ferrous pigmentation. If the disease remains limited to this stage, disability may remain limited being merely due to increased work of breathing.

With extension of the process three major complications arise. There may be progressive invasion of venous adventitia with perivascular fibrosis, intimal hyperplasia, and ultimately occlusion of the vascular lumen. Relatively large venous channels may become involved but arteries tend to escape early damage. This obstruction to the venous channels leads to progressive elevation of pulmonary tension and marked cor pulmonale. Heart failure is the usual cause of death in these cases. The asbestotic origin of the venous obstruction is readily proven by demonstrating the asbestos fibers in the peri- and endovascular granulation tissue. Perl's stain often is sufficient for this purpose as it will demonstrate the presence of iron deposits in the protein sheaths around the asbestos fibers. Another tell-tale feature consists of the deposition of abundant perivascular elastic laminae and fibers.

While the stage of the alveolar mural invasion is not radiographically detectable, except for blurring of lesser pulmonary markings, the stage of perivenous fibrosis is characterized on the *x*-ray by the development of a coarse web and the effacement of the normal vascular pattern. The

extent of the latter damage is often underestimated until tomography or angiography are resorted to.

Massive fibrosis tends to develop mainly in the parts of the lung which are in constant agitation, such as the lung apex, the supradiaphragmatic zone of the basal lobes, and the para-cardiac components. The consolidation is effected by progressive interstitial invasion, atelectasis, trapping of fibers within alveolar spaces where phagocytes surround them and organization takes place, and by abundant pleural thickening on both sides of the lamina elastica. In these zones carnification is often so complete that all semblance of pulmonary architectonics is completely lost. Major blood vessels can only be identified by elastic stains which reveal their elastic skeletons.

Radiographically, these consolidated zones present as diffuse and spreading opacities along the upper mediastinum, above the diaphragm and on either side of the heart, whose silhouette becomes blurred: the so-called "shaggy heart" feature. Calcification may occur in these dense zones.

Symptomatically, these patients usually are merely dyspnoeic on exertion unless there is marked accompanying vascular obstruction. In such cases cor pulmonale will be the predominating lesion. The thorax is usually small and immobile while breath sounds, vocal fremitus and resonance are markedly impaired over the affected zones. Bronchitis and bronchiolitis are not common in asbestosis, though some of these air passages may become involved in the fibro-granulomatous reaction. Advanced cystic bronchiectasis may develop as a result of the fibrotic distortion of the lung. At first there is mere bronchial distension and some fluid or mucus may become trapped in the dilated ducts. Later infection supervenes with all the attendant inflammatory processes leading to chronic suppurative bronchiectasis. The symptoms in these cases do not differ much from conventional varieties of bronchiectasis except that hemoptysis is less common and cor pulmonale develops early.

Tuberculosis may occur in asbestotic subjects, but is not more prevalent among these than it is in the general population. The lesions are not any more severe than those usually seen and often are limited by the fibrous barriers caused by the asbestosis.

Pulmonary carcinoma has been observed with such high frequency in employees of the asbestos industry that a causal relationship has been accepted by most authorities. Indeed, pulmonary carcinoma is compensable as an occupational disease in England, Germany and South Africa. Most of the carcinomata are squamous cell epitheliomata. Pleural mesothesioloma is also quite prevalent, especially in crocidolite industries.

Since the lymphatic system is not completely destroyed in asbestosis, a neoplasm may spread diffusely throughout the lung in spite of extensive interstitial fibrosis. This does not readily occur in silicosis.


A
B

Asbestos bodies not infrequently are demonstrable in the sputum, where they may serve as a diagnostic clue. However, severe asbestosis is compatible with their absence. Again, asbestos bodies may be quite prevalent without coexisting asbestosis. This is possible because the majority of asbestos bodies are formed in the alveolar spaces when fibers have become engulfed by phagocytes. These asbestos bodies may become incarcerated in granulomatous tissue by atelectasis and secondary inflammation.

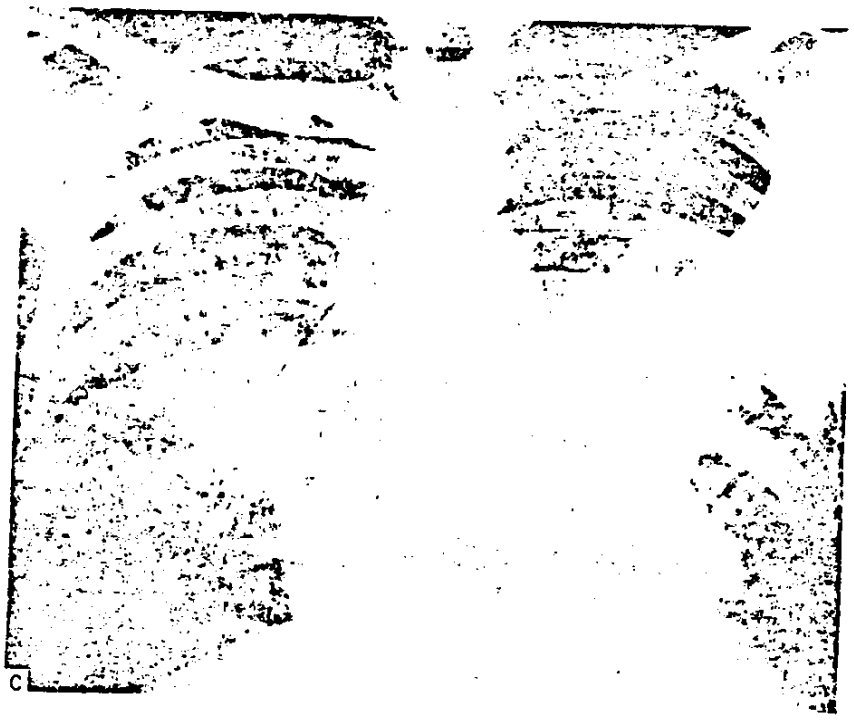

FIG. 57

ASBESTOSIS

RADIOGRAPHIC FEATURES

A, EARLY CHRYSOTILE ASBESTOSIS. Linear pulmonary fibrosis and ground glass opacification. Commencing shrinkage of upper lobes. Incipient cardiomegaly. Diaphragmatic adhesions. ***Textile worker heavily exposed to chrysotile dust 7 years.*** Slight exertional dyspnea and tachycardia. Discontinued work.

B, ADVANCED CHRYSOTILE ASBESTOSIS. Marked interstitial infiltration. Regional opacification. Para-cardiac infiltration and "shaggy heart." Reduced lung volume. Marked cardiomegaly. ***Same case as A, 6 years after discontinuation of exposure to chrysotile dust.*** Marked cardiopulmonary disability. Died less than a year later from advanced chronic cor pulmonale.

C, ADVANCED CROCIDOLITE ASBESTOSIS. Extensive bilateral diffuse infiltration of lung fields. Thickened pleura and subpleural cyst. Part of peribular opacification due to congestive cardiac failure. Heart markedly enlarged. ***Negro miner exposed 6 years to crocidolite dust.*** Marked dyspnea and anasarca. Died two years later from pulmonary carcinoma at the age of 35.

In industries in which asbestos is handled extensively, warts are not uncommonly experienced at points of cutaneous friction, e.g., hands, collar, belt, cuff zones. Many of these acanthotic lesions are indistinguishable from benign warts, but occasionally the tell-tale causative asbestos fibers are identifiable. Later these warts, if left untreated, have a strong tendency to progress to malignant anaplasia. They should, therefore, be radically extirpated as soon as they are identified.

Important features in all cases of asbestosis are the existence of a lag period before the onset of the symptoms and the inexorable progression of the disease despite cessation of dust exposure. There is evidence to support the view that the first few years of exposure to asbestos fibers are as important determinants of the final outcome as are most of the remaining years of exposure. Thus, in one verified case, a single month of exposure to asbestos dust in a textile plant in one country was followed thirty years later in another country by fulminating fatal asbestosis. The intervening period was one of good health and no further asbestos dust exposures had occurred. Continued exposure will, of course, aggravate the process. The severity of the asbestosis is thus a function of the duration of the exposure multiplied by the period which has intervened between the inception of exposure and the onset of the disease. This principle at the same time accounts for the progression of asbestosis after exposure has been discontinued. Cases are currently cropping up in men and women who had worked as pipe insulators on liberty ships during the second world war without since having had any further asbestos exposure.

Talcosis: Two varieties of this condition occur. The first is caused by tremolite tale and the disease is essentially asbestosis with the same range of severe lesions found after chrysotile, amosite or crocidolite exposures. Tale bodies similar to asbestos bodies also occur. This condition should be carefully distinguished from the pulmonary reaction to other forms of commercial tale which may consist of pure magnesium silicate (alpine tale) or may be a mixture of the latter with antigorite, chromite, dolomite, magnesite, and spinell. Most of these substances are relatively inert. When breathed in excessive quantities, and especially when there is some pre-existing pulmonary lesion (e.g. virus pneumonitis), they may cause respiratory incapacitation, usually transient, through extensive alveolar mural cellular infiltration. Because of the multiplicity of uses of tale, such cases may occur in diverse industries where tale is used as a slipping agent (e.g. paper mills, rubber industry). These pathogenic tales have no place as surgical or toiletry accessories.

Rock Wool is a synthetic fibrous silicate extruded from slag. It is extensively used as an insulating material. When its long fibers are inhaled repeatedly and in great quantity, pulmonary lesions closely similar to those seen in asbestosis may result. Once more, the clue to diagnosis is the discovery of "rock wool bodies" in the granulation tissue